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CASE REPORT

A Case of Ischemic Monomelic Neuropathy After Arteriovenous Fistula Placement

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Abstract

Ischemic monomelic neuropathy (IMN) is a relatively uncommon and under-recognized complication of vascular access creation for arteriovenous (AV) fistula in hemodialysis patients. They usually develop distal muscle weakness, sensation loss, and severe acute pain without muscle necrosis soon after AV fistula creation. Physicians should be aware of this condition as prompt diagnosis and timely vascular interventions are necessary to save the limbs and prevent permanent functional disability. Once the diagnosis of IMN is made, the patients will need emergent ligation of the fistula to restore the distal perfusion. We report a case of a 59-year-old male patient with End-stage Renal Disease on hemodialysis who developed severe pain, weakness, and loss of sensation in the left arm a few hours after left brachiocephalic vascular access creation. He was subsequently diagnosed with ischemic monomelic neuropathy and underwent emergent AV fistula ligation. Symptoms were relieved immediately after the ligation.

Keywords: Ischemic monomelic mononeuropathy, Hemodialysis, Arteriovenous fistula, Dialysis access steal syndrome, Hemodialysis access induced distal ischemia

1. Introduction

Ischemic monomelic neuropathy (IMN) can be a debilitating condition in End Stage Renal Disease (ESRD) patients on hemodialysis and develop as a rare complication from under perfusion after vascular surgery for the creation of aterio-venous (AV) fistula in these patients. There are two variants of upper limb ischemia after vascular access surgery or hemodialysis access-induced distal ischemia (HAIDI), including a rare variant, ischemic monomelic neuropathy, and the more commonly encountered dialysis access steal syndrome (DASS).[1] Ischemic neuropathies develop from acute axonal loss in the distal portion of the limb resulting from decreased blood flow in the upper extremity and increased blood flow through the AV fistula after vascular access creation. Other causes for ischemic neuropathies in the upper extremity include thoracic outlet syndrome, trauma, compression from casts or tourniquets, and thromboembolism. Neuropathy commonly occurs in the radial, median, and ulnar nerves. These patients develop neurological deficits with distal muscle weakness, sensation loss, and acute onset of pain immediately after surgery. Symptoms resolve immediately with distal revascularization and ligation of vascular access.

2. Case report

Our patient is a 59-year-old African American male left-hand dominant, has a history of ESRD, and receives outpatient hemodialysis thrice weekly.

Abbreviations: IMN, ischemic monomelic neuropathy; ESKD, end-stage kidney disease; HD, hemodialysis; AV fistula, arteriovenous fistula; HAIDI, hemodialysis access induced distal ischemia; DASS, dialysis access steal syndrome.

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His dialysis access was a tunneled right internal jugular hemodialysis catheter. His other medical comorbidities included essential hypertension, diabetes mellitus type II, nonobstructive coronary artery disease (CAD), benign prostatic hyperplasia, gout, heart failure with preserved ejection fraction, hyperlipidemia, and obstructive sleep apnea. The patient underwent an uneventful elective left brachiocephalic AV fistula creation under the regional inter scalene block on an outpatient basis. But Immediately after the procedure, the patient developed chest pain and shortness of breath for which he was admitted for further evaluation. On examination, the patient was afebrile, blood pressure was 136/90 mmHg, pulse 86/min, Respiratory rate 17/min, and saturating well on the room air. The electrocardiogram showed T wave inversions in the leads I and aVL, as shown below, and an old EKG was not available to compare [Fig. 1]. Serum troponin was slightly elevated at 0.062 (Normal 0.015 ng/ml), with subsequent values trending down to 0.032. His D-dimer was elevated at 1.03 (normal 0.5 mg/L)—other labs as shown in Table 1. A chest X-ray showed no pneumothorax, pleural effusions, or airspace consolidations. The patient was evaluated by cardiology for chest pain and recommended cardiac cath. However, the patient refused the procedure as chest pain had improved. Echocardiogram showed a preserved ejection fraction of 55–60%. A Ventilation-perfusion scan showed a low probability of pulmonary embolism.

Patient started complaining of minimal pain in the left arm after the procedure which progressively worsened to severe pain after a few hours. There was no return of sensation since the procedure and worsening of weakness with decreased strength in his left arm. He could not move his arms and felt his left hand was heavy. The patient developed numbness in the left palmar surface and fingers. On examination, there was no swelling along the left arm or skin changes in the left forearm or wrist. He was found to have a solid left radial pulse, and the left hand was warm to the touch. The area of the AV fistula was sore to the touch without hematoma and swelling. There was a loss of sensation over the palmar aspect of the left hand and all fingers. The range of motion of the left wrist and fingers were reduced with poor wrist and finger extension. The patient exhibited an inability to make a fist and had clawing of the hands. He had motor and sensory deficits in the area of ulnar, median and radial nerve distribution. Overall, there was left upper extremity weakness with 2/5 strength. He had a tunneled dialysis catheter on the right internal jugular vein. The rest of the physical examination was normal. Doppler ultrasound of the left arm showed normal arterial pressures. Differential diagnosis at that time was prolonged nerve block versus ischemic monomelic neuropathy. Our patient had an interscalene block for anesthesia which usually covers most of the brachial plexus and spares the ulnar nerve. However, he developed motor and sensory deficits along the ulnar nerve that ruled out prolonged nerve block effects. He underwent emergent AV fistula ligation as there was a high suspicion of ischemic monomelic neuropathy given the acute onset of symptoms immediately after AV fistula placement, the presence of a strong, palpable radial pulse, and the absence of findings suggestive of tissue ischemia. A nerve conduction study and electromyogram were not done as the patient had to undergo emergent surgery, and also IMN is a clinical diagnosis. Left arm motor strength improved with the ligation. Three days later, the patient

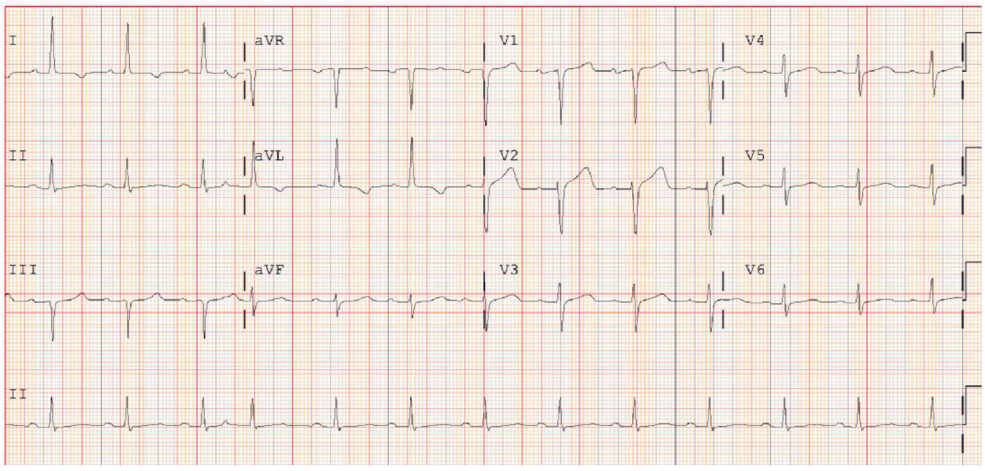

Fig. 1. EKG shows T wave inversions in Lead 1 and AVL.

Table 1.

Labs	Values on the day of admission	Reference Range
WBC	6.44 × 1000/microliter	4–11 × 1000/microliter
RBC	4.13 million/microliter	4.7–6.1 million/microliter
Hemoglobin	10.7 g/dL (gm/dl)	12.5–14.5 gm/dl
Hematocrit	36%	39–51%
Platelet counts	200 × 1000/microliter	150–450 × 1000/microliter
Sodium	137 milliequivalent/Liter (meq/L)	135–145 meq/L
Potassium	4.7 meq/L	3.5–5.1 meq/L
Chloride	104 meq/L	98–106 meq/L
Co2	24 meq/L	22 meq/L
Anion gap	9 meq/L	10–12 meq/L
Glucose	169 mg/dL (mg/dl)	70–105 mg/dl
BUN	30 mg/dl	8–24 mg/dl
Creatinine	5.9 mg/dl	0.7–1.3 mg/dl
GFR	10	>90
Calcium	8.6 mg/dl	8.8–10.2 mg/dl
Total Bilirubin	0.3 mg/dl	0.1–1.2 mg/dl
AST	10 Units/Liter (U/L)	8–33 U/L
ALT	12 U/L	4–36 U/L
ALP	81 International Units/L	44–147 IU/L
Magnesium	2.1 mg/dl	1.7–2.2 mg/dl

underwent a successful open thrombectomy and fistulogram, which revealed a strong thrill, strong doppler signal, and retained palpable radial pulse after the procedure. The patient followed up in 3 weeks and was noted to have a good radial pulse without residual weakness and pain in the arm. The capillary refill was preserved and normal 1 s.

3. Discussion

As the world population ages, the number of patients with CKD increases, and so does the ESRD population. AV fistula is always preferable among the three types of dialysis access because of the least chances of infection and better longevity. As of 2020, The percentage of ESRD patients in the US using an AV fistula is at 63.7%, while the use of an AV graft is at 16.9%, and with catheter use, it is at 19.4%.[2] The commonly encountered complications of vascular access creation can be categorized into mechanical and hemodynamic complications. Mechanical complications include wall degeneration and pseudoaneurysm, which may develop from a puncture hematoma, or infection. Hemodynamic changes related problems could be venous hypertension, arterial steal syndrome, and high-output cardiac failure. One rare hemodynamic mediated complication is IMN.[3]

Bolton et al. in 1979 first discussed distal ischemic neuropathy following a bovine arteriovenous shunt.[4] Terminology IMN was first reported in clinical literature by AJ Wilbourn et al. in 1983.[5] IMN is a variant of hemodialysis access-induced distal ischemia (HAIDI), which can cause distal axonopathy or multiple axonal loss involving both motor and sensory branches of the multiple distal nerves and it develops when the blood flow decreases in the extremity after vascular access creation because of the shunting of blood away from the major arteries. Data on Incidence is limited given its rare presentation and underreported because of unawareness of the condition among the physicians, but estimated to be 0.1% as per Thermann et al.[6] In an extensive review of more than five thousand procedures, Zanow reported the incidence of access-related ischemia to be 1.8% for elbow fistulas and 0.3% for wrist fistulas. Although, the true Incidence of it is unknown.[7] There are no predicting factors for IMN however, for unknown reasons, this injury is more commonly present in females compared to males and diabetes patients.[8] IMN is a rare complication because of the rich collateral arterial blood supply to vasa nervorum. Nerve ischemia occurs because of under-perfusion of vasa vasorum. It is predominantly seen in the brachial artery fistulas as the brachial artery is the only blood supply to the forearm and hand.[9]

Symptoms of IMN can vary from paresthesia, pain, and numbness along the course of distribution of all three forearm nerves, plus or minus diffuse motor weakness or paralysis. The affected hand stays warm, and we can often get a palpable radial pulse or audible Doppler signal. Nerve conduction studies reveal damage to axons, low amplitude or absent response to the motor and sensory nerve stimulation with decreased sensory and motor nerve conduction velocities in the median, ulnar and radial nerves.[10] The presentation of IMN can

mimic axillary block anesthesia neurologic complications, vascular steal, other peripheral nerve compression, carpal tunnel syndrome, and functional deficit secondary to surgical trauma or postoperative pain.[1]

Differential diagnoses of those developing arm weakness, sensation loss, and severe pain after the vascular access creation include dialysis access steal syndrome (DASS), external compression from hematoma, and ischemic monomelic neuropathy. Motor and sensory symptoms can be overshadowed by severe, intractable neuropathic pain. Diagnosis of IMN can be difficult because of its infrequent occurrence and absence of significant tissue ischemia in the involved extremity; however, it can be established based on clinical findings.

There are only three possibilities in our case based on the symptoms and clinical findings which include nerve injury from prolonged anesthesia drug related nerve blocking effect and the two HAIDI variants, IMN and DASS. Our patient had an interscalene block for anesthesia which usually covers most of the brachial plexus and spares the ulnar nerve. However, our patient developed motor and sensory deficits along the ulnar nerve that ruled out prolonged nerve block effects. Differentiating IMN in the upper extremities from DASS is crucial for the execution of appropriate management. Distinguishing features of IMN are rapid onset after the procedure, a palpable radial pulse, and warm upper extremity as it solely involves nerves. In contrast, DASS can be diagnosed by the absence or weak radial pulse, delay in capillary refill, discoloration of fingers, cool hands, symptoms improve with compression of AV access, and skin and muscles are involved more than nerves.[11] Our patient had strong radial pulse, no skin findings, hand was warm to touch and symptoms abated immediately after ligation of fistula Thus, conglomeration of all the symptoms, clinical findings and response to treatment suggestive of IMN.

Limited data is available in the literature on the management as there are no large studies done because of its rare presentation. IMN can cause irreversible neurological deficits and requires immediate AV closure. Out of 19 cases reported by Han et al. from Korea, ten patients (roughly 50%) did not have an improvement in symptoms even after rectifying hemodialysis access.[12] Delay in establishing distal perfusion can cause poor long-term prognosis and worse outcomes with permanent disability or limb loss. These patients should be observed closely after the procedure for any neurological symptoms in the extremities. Even with timely diagnosis and ligation of AV fistula, clinical recovery is variable and partial in most cases, with persistent and significant deficits requiring rehabilitation. Various reports have been published to demonstrate that despite aggressive management, IMN symptoms may be partially reversible and can stay permanent.[13] Only a few case reports are available in the literature that shows complete recovery after ligation.

Thankfully, our patient did recover his symptoms with timely intervention and is doing well currently without residual weakness. These patients will eventually require new AV access at other sites. However, by all practical means, many patients may not have alternative sites for future hemodialysis access construction due to poor vasculature. This complication can occur in other sites too, as the underlying risk factor of the patient stays unaltered. Most patients will be left with residual neurologic impairment. Other treatment measures for IMN are thus mainly conservative, among which an important aspect is pain control. Antidepressants, narcotics, and anticonvulsants have been attempted for pain management.[14]

4. Conclusion

IMN is a rare complication of AV fistula creation which physicians need to be aware of as early recognition and treatment are crucial in saving the limb and preventing disability. IMN is less commonly associated with tissue ischemia and more with nerve injury. The differentials and the presentations can be varied. Our case is unique in the sense that it was diagnosed very early into its course and timely interventions helped salvage the limb with no residual neuro deficits. A global awareness among the multidisciplinary team, including nephrologists, hospitalists, neurologists, vascular surgeons, nurses, interventional radiologists, and dialysis care technicians, is needed to identify and address the early consideration of IMN. More and more cases of IMN are needed to be recorded and published to gain more expertise with management outcomes and to add more literature to the database.

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Informed consent

Verbal consent obtained.

Ethics approval

Our institution does not require ethical approval for reporting individual cases or case series.

Author contributions

VS reviewed patients' charts. VS, MP, NE, NRK, SR and IM contributed in writing introduction, discussion and conclusion. All authors contributed equally to preparation of this manuscript and all of the authors reviewed the manuscript and agreed with the findings and interpretation.

Data availability

From the patient's chart and PUBMED.

Conflict of interest

None.

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